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## *Escherichia coli* Induce Reactive Arthritis in Iraqi Arab Patients

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### Abstract

Reactive arthritis (ReA) is an inflammatory arthritis that arises after certain types of gastrointestinal or genitourinary tract infection. It belong to the group of arthritis known as spondylo arthropatients. Infection is one of the important causes for the inflammation of the joint (arthritis), included bacteria. Seventy five urine sample from Iraqi patients with ReA with age range (20-60) years. Microscopically and culturing for urine was done, *Eschrichia coli* was recorded 24 (32%) , Therefore *Eschrichia coli* may be one of causes of urinary tract infection which induced reactive arthritis and urine examination and culture of urin should be done as routine in diagnosis and reactive arthritis patients.

**Keywords:** Reactive arthritis, UTI, bacteria

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## **1. Introduction**

Reactive arthritis is an inflammatory arthritis [1]. The duration of reactive arthritis is considered to be chronic when it extends over six months [2]. Infection can be triggering factor in some chronic, which affect joint, prolonged inflammation damage joint capsule, auricular cartilage, bone and tendon consequence to a synovial ischemic from markedly increase intra synovial fluid pressure and compression of blood vessel, phloistic product of invading organisms and host defense mechanism [3]. ReA was associated with HLA B-27 [4]. This allele enhance the intracellular survival of microorganism and then arthritogenic or alter the immune response to produce more proinflammatory chemokines [5]. Many cell Gve<sup>-</sup> bacteria can induced ReA, the presence of LPS in joints is a common of pathogenically important feature of ReA [6]. Reactive arthritis following *E. coli* urinary tract infection was very rare [7], urinary tract infection showed be included in diagnostic investigation of patients with acute arthritis [8]. Other research showed that acute bacterial gastrointestinal was associated with higher risk of arthritic symptoms [9].

## **2. Material and methods**

### **2.1 Patients group**

The study was carried out 75 Iraqi Arab reactive arthritis patient's (40female and 35 male) were referred to the consultant clinic at department of rheumatology, Baghdad teaching hospital during period from November 2013 to August 2014, the diagnosis was made by the consultant medical staff at the clinic, according to ACR criteria with aid of laboratory diagnosis (ESR, CRP, RF) .

### **2.2 Control group**

Thirty nine healthy persons who matched with patients for age and gender were selected.

### **2.3 Laboratory methods**

#### **2.3.1 Blood sample collection**

Five ml of blood was draw from each subject (patients and control). 2 ml was put in EDTA tube for ESR evaluation . 3 ml of blood was patient test tube, then centrifuge to collect serum for CRP and RF evaluation. CRP and RF was evaluated semi-quantitively by latex agglutination according to leaflet of company manufactured ( Spin react , Spain).

#### **2.3.2 Urine Samples collection**

The diagnosis of urinary tract pathogens is based on the quantitation of bacteria in the urine. The culture is taken from midstream, clean catch urine specimen. Blood agar, MacConkey agar were used for the culture of urine samples, For isolation of G+ve and G-ve bacteria, or to detect the hemolysis of bacteria if the *E .coli* producer hemolysin. All samples were collected and transported to the laboratory within an hour at low temperature to

inhibits bacterial multiplication in the urine samples until processed in laboratory. This is very important because the number of bacteria in the urine sample can be increased if the urine sample is not stored properly, and then the small number of bacteria may multiply to large numbers and lead to a false positive significant bacteriuria. The urine samples were examined and cultured in less than two hours [10].

### ***2.3.3 Evaluation of urine for bacterial culture.***

Urine Sample was examined by microscope (General urine examination), then it was cultured. 5 ml of urine sample was transferred into test tube centrifuge tube and centrifuged at 3000 rpm for 10 minutes. One drop of the sediment was placed on a glass slide and covered with a cover slip, then it was examined by high power objective lens (40x). Then examined different field for pus cells [11]. The urine was mixed well and the loop was inserted into the urine vertically to allow urine to adhere to the loop. Then a loop full of urine was spread uniformly on the surface of blood agar plate and McConkey agar plate. were incubated for 24 hours at 37°C [12].

### ***2.3.4 Identification of bacteria by auto analysis vitek2 compact device.***

#### ***2.3.4.1 principle***

It was used in industrial microbiology-testing environment include 21 CFR Part 11 compliance (for electronic records and signatures) and a colorimetric reagent card (BCL) used to identify the spore-forming Gram-positive bacilli (i.e., *Bacillus* and related genera). The other colorimetric reagent cards (GN, GP, YST) apply to all system formats for both industrial and clinical laboratories.

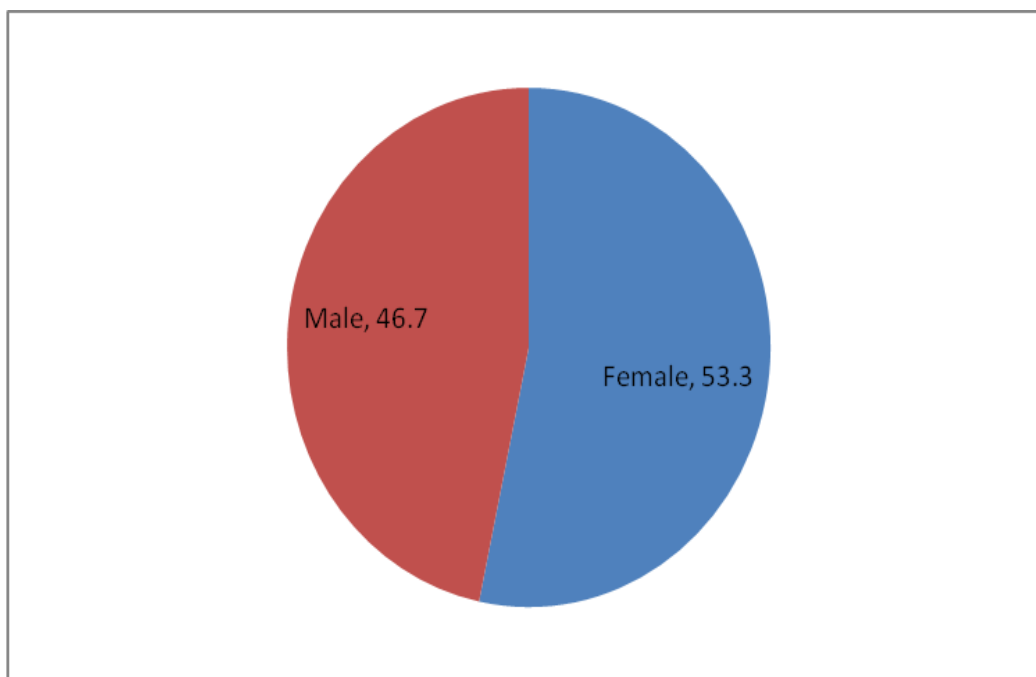
#### ***2.3.4.2 Assay of identification***

The identification system for bacterial isolate (gram negative and gram positive bacteria), and it was done in Al-Shahid Dr. Feroz General hospital. as following:

1. Loop full of Fresh culture after 24hrs (single colony). Put in 3ml of normal saline, shaken and measured the turbidity by turbidity meter.
2. The gram positive card or gram negative card entered in the tube, and put it in the casket place.
3. Insert the casket in the room number 1 of the vitek 2 compact to load bacterial suspension.
4. Moved the casket from room one to room two to begin reading (late for 6 hrs the results appeared on the computer screen as a table contained all details of bacterial isolate).

## **3. Results and discussion**

The present study showed that female 53.3% had the higher percentage than male 46.7% as in fig (1).



**Figure 1:** Sex percentage of reactive arthritis

The mean of age of patients was  $38.1 \pm 1.5$  years in ReA patient while  $39.1 \pm 1.6$  years in control, the high percentage of ReA patient was in 20-40 years (71.8%) with mean of age  $33.6 \pm 1.1$  years in compared within 41-60 years the ReA patient was 28.2% with mean of age  $49.8 \pm 1.6$ . The result was agreed with [13]. The true incidence was under estimated, predominantly affects young adult in the 20 - 40 age group. Other found the children may be affected in addition to people who are 20 - 40 years of age after *Streptococcus* infections [14]. ReA which has single peak incidence at 27 - 34 years [15]. Other showed that reactive arthritis affected a young age group, half the episodes occurred in age range 16 - 24 years in Southern New Zealand [16]. The patients with ReA were adult (age range 40 - 47years) [17].

There was a significant differences ( $P < 0.05$ ) of ESR in ReA ( $70 \pm 2$ ) .mm/hr Than control ( $23 \pm 2$ ).mm/hr. current result disagreed with studies of [18] that was ESR (mm/hr) ( $45.2 \pm 32.2$ ). ESR in ReA was  $> 60$  mm/hr. [19]. the ESR is important in the diagnosis of inflammatory conditions and in the prognosis of non-inflammatory conditions [14].

All patients had seropositive for CRP in compared with RF all patients was seronegative rheumatoid factor. These agreed other studies that was CRP positive and RF negative [20]. CRP are elevated at the onset of disease. Later CRP may become normal in the chronic stage of disease while  $ESR > 60$  mm/hr [21,22]. CRP level serve as a reliable marker of disease activity in rheumatoid arthritis and various vasculitis. CRP does not appear with some autoimmune disease like SLE. CRP is also known to infection disease, it is a marker of infection and participate in host defense, it is a sensitive marker for acute and chronic inflammation [23]. Other study showed that elevated CRP level may be mechanically link to more inflammatory synovial response in disease joint [24], CRP is helpful in determining the presence of septic joint [25].

The result of present study showed that *E. coli* bacteria was recorded higher percentage in ReA, 32%, *Providencia rettgeriis* 4%, mix bacteria 6.7%, *Enterococcus faecalis* 6.7 %, *Micrococcus lyteus* 2.7%, *Alicalgens* 1.3 %, *Staphylococcus hemolytic* 9.3 %, *Kocuria* 2.7%, no growth 24% as shown in figure (2). Other result showed that *E. coli* was one of urinary tract infection (UTI) in reactive arthritis, ReA is associated with intestinal infection and common urinary tract infection [7]. *Providencia rettgeriis* cause UTI and septicemia especially in immune comprised patients [26]. *Enterococcus faecalis* cause many serious human infection, including UTI and bacteremia [27]. The invasion of *E. coli* into urinary epithelium result in expression of variety cytokines, inducing of apoptosis in infected cells [28]. A large number of different gram negative rods acts as infectious agent on joints. The most frequent detected pathogen of joint infectious are *Staphylococcus aureus*, other gram positive agent like *Streptococcus pyogenes*, *Enterococcus faecalis* [29], [30] papered that UTI caused by *Staphylococcus saprophyticus*. *Kocuria* spp. Are etiologically associated with catheter-related bacteria [31], *Kocuria kristinae* was opportunistic pathogen in immunocomprised patients and elderly [32]. Septic arthritis caused by *Aeromonas* was rare [33], *Staphylococcus hominis* and *S. caprae* had been reported in reactive arthritis [34] were reported that antibodies formed against *Proteus mirabilis* was detected in 72.2% of children with marker of ophthalmoarticular rheumatic disease from the above result of urine examination aid the diagnosis of ReA by which bacteria can trigger this disease especially in patients with urinary tract infection .

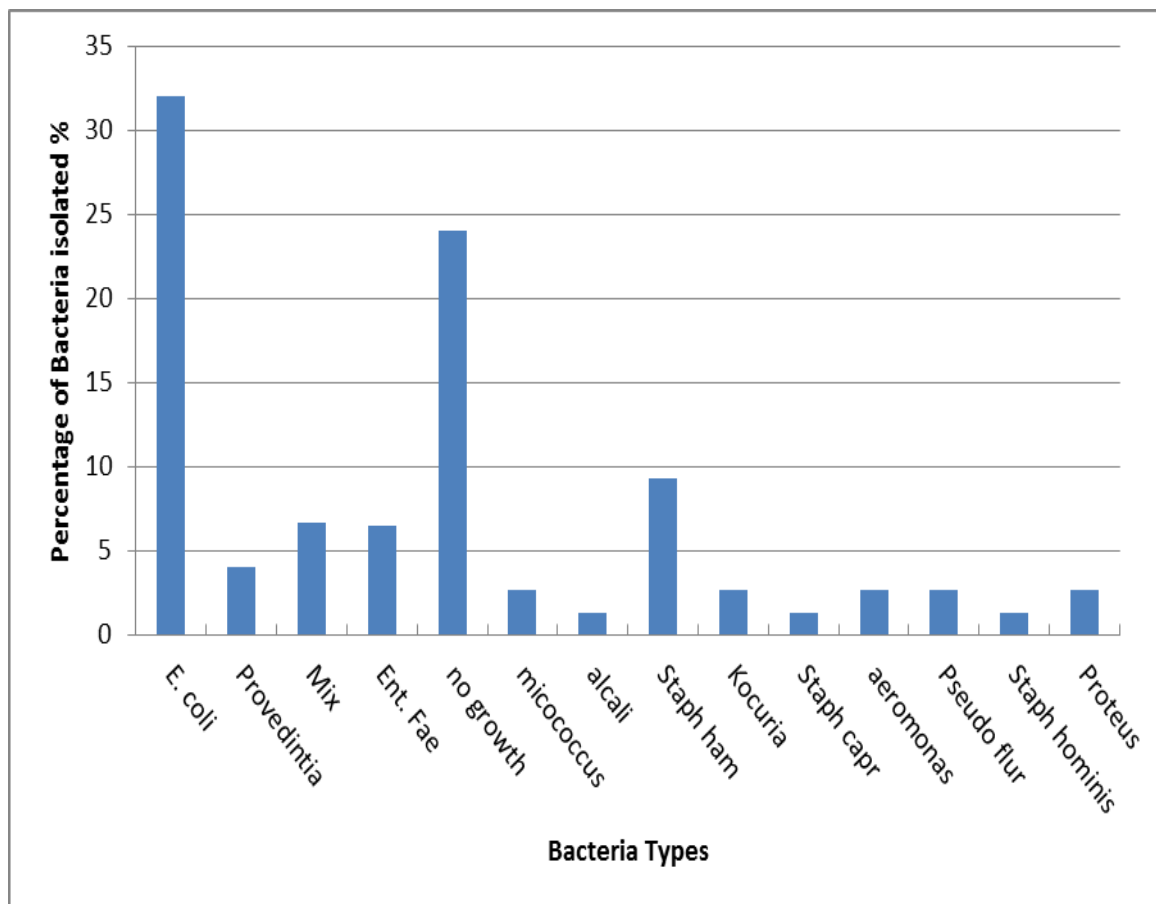


Figure (2): Bacterial frequencies in ReA infected with UTI.

## References

- [1] C. T. Parker and D. Thomas. Reiter's syndrome and reactive arthritis. *J Am Osteopath* .,100(2):101–104. 2000.
- [2] J. Braun ; D. Kingsley ; V. Heijde and J. Sieper. On the difficult of establishing a consensus on the definition of and diagnostic investigations for reactive arthritis. The 4<sup>th</sup> International Workshop on Reactive Arthritis, Berlin, Germany, July 3/6/1999. *J. Rheumatol* , 27(9): 2185-2192. 2000
- [3] J. Sibia and F. Limbach. Reactive arthritis or chronic infectious arthritis. *Ann. Rheum. Dis.*, 61:580-587. 2002.
- [4] I. Colmegna ; R. Ouchacovich and P. Espinozo. HLA B27 associated reactive arthritis. *Clin. Microbiol. Rev.*, 17(2):348-369. 2004.
- [5] K. Kapasi and R. D. Inman. Epitope of HLA B<sub>27</sub> confers class I mediated modulation of gram negative bacteria. *Invasion*, 153:833 – 840. 1994.
- [6] K. Granfors ; S.Jalkanen and O. Maki. *Salmonella* lipopolysaccharide in synovial cell from patients with reactive arthritis. *The Lancet*. 335:685 – 688. 1990.
- [7] F. Renou ; G. Wartel and I. Raffary. Reactive arthritis due to *E. coli*. *Urinary tract infection. Rev. Med. Int.*, 32(1):4 – 6. 2001.
- [8] K. Laasila and R.M. Leirisalo. Recurrent reactive arthritis associated with urinary tract infection by *E. coli*. *J. Rheumatol.*, 26(10):2277 – 2279. 1999.
- [9] L.H. Linda and J.W. Freston. "Diseases: Manifestations and Pathophysiology". in: *Remington Pharmaceutical Sciences*. 16<sup>th</sup> edition, Pennsylvania: Mack Publishing Company, PP: 615. 1980.
- [10] M. Cheesbrough. Medical laboratory manual for tropical countries. *Microbiology*. 2: 400-480. 1985.
- [11] W.E. Stamm. Measurement of pyuria and its relation to bacteriuria. *Am J Med.*, 75(1B):53-58. 1983.
- [12] S.S. Hamdulay ; S.J. Glynne ; A. Keat. When is arthritis reactive?. *Postgrad Med J.*, 82(969):446-453. 2006.
- [13] Y. Uziel ; L. perl ; J. Barash and P.J. Hashkes. Post streptococcus reactive arthritis in children a distinct entity from acute rheumatic fever. *Pediatr Rheumatol Online J.*, 9:32-34. 2011.
- [14] S.L. Mackie ; A. Keat. Poststreptococcal reactive arthritis: what is it and how do we know?. *Rheumatology*. 43(8):949-954. 2004.
- [15] J. Highton and D. Priest. Reactive arthritis: characteristics in southern New Zealand. *N Z Med J.*, 109(1018):93-95. 1996.
- [16] T.H. Mathila ; J.P. Nuorti ;P. Rutu ; J. Mikkola ; A. Sitonen and M. Leivisalo. Reactive arthritis after an outbreak of *Yersinia pseudotuberculosis* serotype 0:3 infection. *Ann Rheum Dis.*, 62:866-869. 2003.
- [17] J.M. Van Bommel ; V. Delgado. ; E.R. Holman ; C.F. Allaart ; T.W. Huizinga; J.J. Bax and A.H. Van der Helm-VanMil. No increased risk of valvular heart disease in adult poststreptococcal reactive arthritis. *Arthritis Rheum.*, 60(4):987-993. 2009.
- [18] H.M.M. Van den Hoogen ; B.W. Koes ; J.T.M. Van Eijk and L.M. Bouter. On the accuracy of history, physical examination, and erythrocyte sedimentation rate in diagnosing low back pain in general practice. *Spine* . 20(3):318-327. 1995.

- [19] A. Dimitrova ; V. Valtchev ; I. Yordanova ; H. Haidudova ; D. Gospodinova and S. Tisheva. Keratoderma blenorrhagicum in a patient with Reiter Syndrome . *JIMAB* . 14: 68-69. 2008.
- [20] S. Mukherjee and M. Kar. Reactive arthritis: current perspective. *Indian Academy clinical Medien.*, 1(3):234-239. 2000.
- [21] T.W. Duclose. C-reactive protein as a regulator of autoimmunity and inflammation. *Arthritis and Rheumatism*. 48(6):1475-1477. 2003.
- [22] A. B. Pearle ; C. R. Scanzuso ; S. S. George and L. Mandl. Elevated CRP level in osteoarthritis are associated with local joint inflammation. 51<sup>st</sup> Annual Meeting of Orthopaedic research Soc. Poster :1412. 2004.
- [23] A. A. Emst; S. J. Weiss; L. A. Tracy and N. R. Weiss. Usefulness of CRP and ESR in predicting septic joint. *Spouth Med J.*, 103(6):522-526. 2010.
- [24] T. E. W. Feltkamp. Factors involved in the pathogenesis of HLA-B27 associated arthritis. *Scand J. Rheumatol.*, 24(Suppl. 101):213–217. 1995.
- [25] H. J. Cho; S. J. Cim; S. Y. Chun and K. O. Park. *Providencia rettgi*. *Infect Chemotherp.*, 42(6): 428 – 430. 2010.
- [26] Huycke, M.M. Sahm, D. F. and Gilmore, M.S. (1998). Multiple drug resistant enterococci: The nature of problem and agenda for future. *Enrg. Infect. Dis.*, 4: 239 – 249.
- [27] J. D. Schilling; M. A. Mulveg; R. G. Vincent and S. J. HuHgren. Bacterial invasion augments epithelial cytokine response to *E. coli* through LPS. *J. Immunol.*, 166: 1148 – 1155. 2001.
- [28] U. Geipel. Pathogenic organism in hip joint infections. *Int. Med. Sci.*, 6(5): 234 – 240. 2009.
- [29] B. A. Gunn and J. C. Davis. *Staphylococcus haemolyticus* urinary tract infection in a male patients. *J. Clin. Microbiol.*, 26(5): 1055 – 1057. 1999.
- [30] S. M. Edmond; C.L. Wong; K. Lali; W. C. Yam and C. W. Angus. *Kocuria kristinae* infection associated with acute cholecystist. *Infectious Dis.*, 5: 60 – 68. 2005.
- [31] M. Mutlu; Y. Aslan; D. Gokdogan; L. Surer and F. Aktag. Septic arthritis caused by *Kocuria kristinae* in a newborn. *HKJ paediatr*, 18:223 – 225. 2013.
- [32] D. D. Ouedraogo; J. Tiendrebeogo; H. Tieno; I. Diallo; A. Bagbila and J. Drabo. *Aeromonas hydrophila* septic arthritis in patients infected with HIV. *Open J. Rheumatol. and autoimmune*, 3(3): 2 – 10. 2013.
- [33] J. A. Wright and S. P. Nair. Interaction of Staphylococci with bone. *Int. J. med. Microbiol.*, 300(2 – 3): 193 – 204, 2010.
- [34] L. A. Katargina; A. V. Starkova and N. E. Iasterbova. Clinical and pathologic significance of *Proteus irabilis* antibodies in uveitis associated with joint lesion in children and adolescent. *Vesofthamol.*, 118(3): 28 – 30, 2002.